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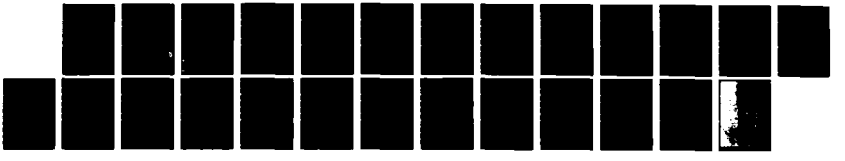

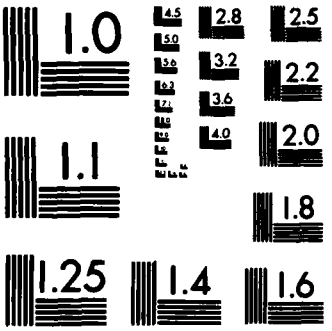

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This study was designed to assess the effects of hypohydration (-5% body weight) and heat acclimation on plasma cortisol and growth hormone responses to exercise (1.34 m/sec^{-1}) in a thermoneutral (20°C, 40%rh), hot-wet (35°C, 79% rh), or hot-dry (49°C, 20%rh) environment. Exercise in a thermoneutral environment while euhydrated resulted in significant ($p \leq .05$) decrements in plasma cortisol (PC); however, when hypohydrated, PC levels were significantly ($p < .02$) elevated. Acclimation attenuated the PC elevations elicited by hypohydration. Hypohydration also effected significant ($p < .02$) increases in growth hormone

(GH) in the thermoneutral environment. Exercise in a hot-wet or hot-dry environment resulted in significant ($p<.05$) increments in both hormones when hypohydrated; these effects were, in several instances, attenuated ($p<.05$) by acclimation, particularly in the hot-wet environment. We concluded that the PC and GH responses were amplified when subjects were hypohydrated and, in several instances, these responses were attenuated by heat acclimation. Additionally, our data indicated that gender does not affect the direction or intensity of these hormonal responses.

Hypohydration and acclimation: effects on hormone responses to exercise/heat stress

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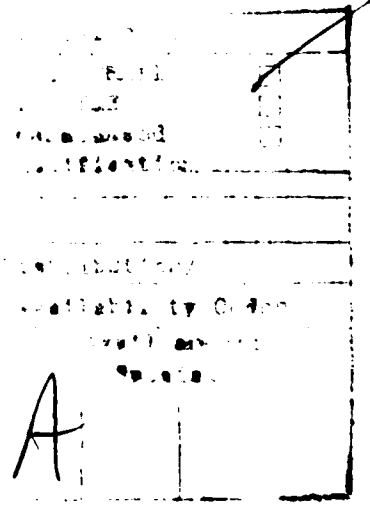

Abstract

This study was designed to assess the effects of hypohydration (-5% body weight) and heat acclimation on plasma cortisol and growth hormone responses to exercise (1.34 $m \cdot sec^{-1}$) in a thermoneutral (20°C, 40% rh), hot-wet (35°C, 79% rh), or hot-dry (49°C, 20% rh) environment. Exercise in a thermoneutral environment while euhydrated resulted in significant ($p < .05$) decrements in plasma cortisol (PC); however, when hypohydrated, PC levels were significantly ($p < .02$) elevated. Acclimation attenuated the PC elevations elicited by hypohydration. Hypohydration also effected significant ($p < .02$) increases in growth hormone levels (GH) in the thermoneutral environment. Exercise in a hot-wet or hot-dry environment resulted in significant ($p < .05$) increments in both hormones when hypohydrated; these effects were, in several instances, attenuated ($p < .05$) by acclimation, particularly in the hot-wet environment. We concluded that the PC and GH responses were amplified when subjects were hypohydrated and, in several instances, these responses were attenuated by heat acclimation. Additionally, our data indicated that gender does not affect the direction or intensity of these hormonal responses.

Key words: Cortisol, growth hormone, exercise, sex differences

Introduction

In their early review on human endocrinological responses to environmental stress, Collins and Weiner (3) concluded that adrenocortical activity increased during heat stress, but duly noted the inadequacy of extant assay techniques. These authors also reported (3) that there was no direct evidence to indicate adrenocortical alterations in the long-term adaptation to heat exposure. Much later, Cochrane et al. (2) reported that following heat acclimatization plasma cortisol levels were reduced, and were unaffected by salt loading. In a more detailed report the same group (4) noted that while plasma renin activity and aldosterone were unaffected by acclimation, plasma cotisol levels were slightly reduced by heat acclimation. From these earlier studies it might be concluded that the intensity of the physiological and perceived stress accompanying passive heat exposure or exercise in a hot environment may be attenuated by the acclimation process. This hypothesis was corroborated by the work of Follenius et al. (7) who reported that increments in plasma cortisol occurred only in those heat-stressed subjects who reported physical discomfort during the heat stress. Even upon exposure of subjects to 100°C ambient conditions Leppaluoto et al. (14) demonstrated inconsistent effects on plasma adrenocorticotrophic hormone, which they attributed to the fact that their subjects were accustomed to such temperatures as sauna devotees.

Nonetheless, in the same report this group demonstrated (14) that plasma levels of growth hormone rose by approximately 250% despite the habituation of their subjects. In an earlier study, Okada et al. (18) reported that exposure of male subjects to 48°C resulted in significantly elevated growth hormone levels while plasma cortisol was unaffected. While Brown et al. (1) have reported that in primates growth hormone responses to stress may be attenuated by adaptation, no such studies have been accomplished using human volunteers.

Thus, while several reports have assessed the role of fluid regulatory hormones (5,6,11) in the process of heat acclimation, very few have examined the response of stress hormones, particularly with the more sensitive assay techniques currently available. Therefore, we have investigated the effects of heat acclimation on the magnitude of the stress hormone response during exercise in a hot-wet or hot-dry environment. In addition, since hypohydration has been reported to increase the level of the physiological strain during heat exposure or exercise in the heat (16, 17), we have also examined and documented the effects of hypohydration on heat/exercise-induced stress hormone response; heretofore, the effects of hypohydration on plasma cortisol and growth hormone responses to heat/exercise stress had not been assessed. Moreover, the availability of a dedicated group of female volunteers permitted us to determine whether gender mediates these hormonal responses.

Methods

Eight male (23.6 $\pm$ 2.8 yrs, 170.8 $\pm$ 7.2 cm, 75.4 $\pm$ 7.4 kg, mean age, height, weight $\pm$ SD, respectively) and 8 female (25.0 $\pm$ 4.2 yrs, 163.0 $\pm$ 6.6 cm, 62.2 $\pm$ 11.3 kg) test volunteers (Ss) participated in this study after being fully apprised of its rationale, methods, procedures, and potential risks. Ss reserved the right to withdraw from the study at any time without retribution.

A total of 12 tests were completed, 6 before and 6 after a period of heat acclimation. Thus, prior to and following acclimation Ss underwent two experimental tests in each of three environments. The three environments selected were: thermoneutral (Ta = 20^{o}C, rh = 40%), hot-wet (Ta = 35^{o}C, rh = 79%), and hot-dry (Ta = 49^{o}C, rh = 20%). The test protocol was repeated in each environment -once when Ss were euhydrated and once when hypohydrated. Hypohydration was achieved by voluntarily reducing fluid consumption for 24 h

before a test and also by performing light exercise in a hot (Ta = 38°C, rh = 20%) environment until each volunteer lost 5% of his or her initial body weight. When the appropriate weight loss was attained, Ss were removed to a comfortable environment and spent the night under supervision. Subjects arose at 0600 h, were weighed, ate a light breakfast if their weight remained sufficiently low, and experimentation began at approximately 0800 h.

Each test protocol lasted for a total of 140 min (4 repeated intervals of 10 min rest and 25 min exercise). Exercise was performed on a level treadmill at 1.34 $m \cdot sec^{-1}$, and during each rest period Ss were weighed and cool tap water was ingested to maintain either baseline weight or -5% from baseline in the case of the hypohydration experiment.

Heat acclimation was achieved by walking on a level treadmill at 1.34 $m \cdot sec^{-1}$ for two 50 min exercise periods interrupted by 10 min rest. This procedure was repeated for 10 consecutive days with alternate hot-wet and hot-dry conditions (as noted above). During acclimation and test intervals, Ss wore shorts, t-shirts, and tennis shoes; ad lib water was available during the acclimation regimen, and in quantities sufficient to maintain starting weight on test days.

Blood samples (5 ml) were taken at intervals on each of the experimental test days; blood was obtained from catheters placed in superficial arm veins. The first blood specimen (time 0) was taken after Ss remained in a standing position for 20 min in a comfortable environment (Ta = 20°C, rh = 30%). The second (time 1) and third (time 2) samples were removed at approximately 15-20 min into the first and second exercise interval in each environment. The final blood specimen (time 4) was taken upon termination of the fourth exercise interval or when exercise ended if the S could not complete the entire protocol. Following collection, the blood was transferred to heparinized tubes, centrifuged (10000 g, 4°C), and the plasma frozen (-20°C) for subsequent assay.

Plasma samples were thawed and levels of cortisol (PC) and growth hormone (GH) were quantitated by radioimmunoassay techniques. PC radioimmunoassay test kits were purchased from Damon Diagnostics (Needham, MA), and the assay was performed by procedures outlined in their technical bulletin. GH was also measured using commercially available test kits manufactured by International CIS (Saluggia, Italy) and distributed by Damon Diagnostics. Again, the procedures used were as outlined in their technical bulletin.

Statistical analyses were performed by means of Student's t test on the appropriate paired samples. When values at each of the time intervals were compared with the control or pre-exercise (time 0) value, then Dunnett's t test (15) was used. The null hypothesis was rejected at $p < .05$.

Results

Fig. 1 demonstrates the effects of hypohydration and acclimation on levels of PC during exercise in a thermoneutral environment. In the pre-acclimation test, hypohydration resulted in significantly increased levels of PC at time 0 ($p < .02$), time 1 ($p < .05$), time 2 ($p < .025$), and time 4 ($p < .005$). Examining the effects of exercise itself, we observed a significant ($p < .05$) decrement in the euhydrated condition (time 0 vs time 2 or time 4). Although a similar trend was observed when hypohydrated, significance was not achieved. Following acclimation, hypohydration had no effects ($p > .05$) on levels of PC. Again, however, in both the euhydrated (time 0 vs times 2,4 - $p < .05$) and hypohydrated (time 0 vs times 1,2,4 $p < .05$) conditions, there occurred significant reductions in levels of PC during exercise. While acclimation had no effects on PC levels when Ss were euhydrated, there occurred significant decrements during hypohydration (pre-acclimation vs post-acclimation, hypohydration, time 2 - $p < .05$; time 4 -$p < .001$).

Fig. 2 illustrates the effects of hypohydration and acclimation on levels of PC during exercise in a hot-wet environment. Pre-acclimation, hypohydration (vs euhydration) significantly increased PC levels at time 2 ($p < .05$) and time 4 ($p < .001$). Exercise induced a significant increment only in the hypohydrated condition (pre-acclimation, hypohydration, time 0 vs time 4, $p < .05$). Following acclimation, there again occurred a significant ($p < .001$) increment in the hypohydrated state (vs euhydrated, time 4) although no significance was observed at the earlier time intervals. Exercise resulted in a significant increment again only in the hypohydrated condition and only in the time 4 (vs time 0, $p < .05$) sample. Finally, the effects of acclimation (pre vs post) were noted in a significant ($p < .005$) decrement in the hypohydrated condition (time 4).

Fig. 3 illustrates the effects of hypohydration and acclimation on PC responses during exercise in a hot-dry environment. In the pre-acclimation trials hypohydration (vs euhydration) significantly ($p < .005$) increased PC (time 4). This elevation also resulted in a significant ($p < .05$) increase during exercise (time 0 vs time 4) in the hypohydration test. Following acclimation, the effects of hypohydration (vs euhydration) were manifested in significant ($p < .001$) increments in the times 2 and 4 samples. It is also notable that following exercise there occurred significant ($p < .05$) decrements in PC levels in the euhydrated state (time 0 vs times 2 and 4). When hypohydrated, however, exercise in the hot-dry environment elicited a significant ($p < .05$) elevation in PC level (time 0 vs time 4, $p < .05$). In this environment no significant effects of acclimation were observed in either the eu- or hypohydrated state.

Fig. 4 depicts the effects of acclimation and hypohydration on plasma GH responses during exercise in a comfortable environment. Pre-acclimation there were significant increments in GH in the hypohydrated state at time 0 ($p < .025$) and time 1 ($p < .02$); however, these effects were not evident at times 2 and 4.

Exercise or acclimation had no further significant effects on GH levels in this comfortable environment.

In the hot-wet environment (Fig. 5), pre-acclimation, there occurred a significant increment in the hypohydrated condition (vs euhydrated, time 1, $p < .05$). When hypohydrated, exercise resulted in significant ($p < .05$) increments at times 1 and 2 (vs time 0), while a single (time 0 vs time 2) increase occurred when euhydrated. Following acclimation, however, the effects of both exercise and hypohydration were neutralized. Acclimation (post vs pre) resulted in significant decrements in the euhydrated state (time 4, $p < .02$) and the hypohydrated condition (time 2, $p < .01$).

Fig. 6 demonstrates the effects of acclimation and hypohydration on plasma GH levels during exercise in a hot-dry environment. Pre-acclimation, hypohydration significantly ($p < .01$) increased GH levels at times 0 and 1 while exercise elicited a significant increase in a single (time 0 vs time 2, $p < .05$) sample in the euhydrated state. Subsequent to acclimation there persisted a significant ($p < .02$) increase in GH levels while hypohydrated (time 1), but exercise did not affect GH concentration in either the euhydrated or hypohydrated condition. Heat acclimation (post vs pre) had no effects on GH levels either while eu- or hypohydrated in this hot-dry environment.

Discussion

It is noteworthy that no differences in response patterns occurred between male and female test volunteers; therefore, the data were combined for graphic and statistical purposes. The design of the present study, as well as the large number of samples processed, permitted us to draw conclusions about the effects of hypohydration, heat acclimation, gender, and heat-exercise stress on the hormones under consideration. We found this comprehensive approach to be

useful in determining the comparative levels of stress generated by the physiological (hypohydration), physical (exercise), and environmental (hot-wet or hot-dry) variables. Further, the mitigating effects of acclimation were also evaluated by changes in the magnitude of the hormonal responses.

Prior to heat acclimation, hypohydration generally resulted in increased circulating levels of PC, and these increments were exacerbated following exercise in either of the hot environments. Subsequent to acclimation, the effects of hypohydration were moderated in several instances indicating that the process of heat acclimation reduced the physiological effects of these combined stressors. During the light exercise in the thermoneutral environment, PC levels were generally reduced. These reductions may be partially attributable to the hemodilution accompanying mild exercise in this comfortable environment (20) and the normal circadian reduction in PC usually occurring between the morning to noon hours (12). It should be noted, however, that the reductions noted above in the thermoneutral environment do not occur in either stressful environment. When euhydrated, neither the hot-wet nor hot-dry environment was sufficiently stressful to induce an adrenocortical response either before or after acclimation.

The incremental effects of hypohydration on plasma levels of these stress hormones generally reflected the increased physiological stress manifested in increased rectal temperature and heart rate and decreased sweat rate (19). Gaebelein and Senay (9) also demonstrated an increased physiological cost (heart rate and rectal temperature) of both cycling and block stepping after a period of fluid deprivation. In an earlier study Francis (8) reported that when men were exercised in a warm, humid environment, indices of stress (plasma cortisol, dopamine-B-hydroxylase, uric acid) were markedly increased when Ss received no fluid supplement. In the current study it is noteworthy that at several sampling intervals the process of heat acclimation evidently reduced the strain of

hypohydration as manifested in attenuated increments in PC responses. This observation may be compatible with the increased plasma volume associated with heat acclimation (21), assuming that the increased plasma volume would permit an elevated sweat rate and more efficient thermoregulation.

In considering the responses of circulating GH levels to exercise in the thermoneutral and hot environments, there again occurred several increments when Ss were hypohydrated. Once again, several of these elevations were moderated by the process of heat acclimation. It should be recalled that a great deal of interindividual variation occurs among individuals in the response of GH to various stressful stimuli (18). This is mostly true because of the wide variability among individuals in the episodic pattern of growth hormone secretion (22). Thus, the magnitude of standard deviations will, in many instances, preclude statistical significance although apparent trends may be evident.

To the best of our knowledge the effects of acclimation and hypohydration on plasma GH responses to exercise in the heat have not been addressed previously. In fact, only several earlier studies have addressed the response of growth hormone to heat or exercise stress. Kuoppasalmi et al. (13) demonstrated significant effects of strenuous anaerobic exercise on GH while Okada et al. (18) reported the effects of passive heat exposure. As in our own experiments several earlier studies (13, 22) demonstrated a dissociation between PC and GH responses during the imposition of stressful stimuli; Brown et al. (1) noted a similar dissociation in another primate, the squirrel monkey. Clearly, the diverse PC-GH responses noted in our experiments preclude generalizations concerning hypophseal-adrenocortical activity.

It is interesting to note from the current data that irrespective of acclimation level, hydration state, or environment, GH concentrations generally peaked at the mid-exercise sampling times and were ordinarily reduced at the

completion of exercise. These data are analogous to those of Hartley et al. (10) who also demonstrated, before training, increments in plasma GH levels during exercise; at exhaustion, however, these levels had fallen considerably. Following physical training, these authors (10) attributed a persistent GH elevation during exercise to an endocrinological adaptation designed to provide additional oxidizable substrates. In the current experiments, however, training effects were negligible, and the reductions noted toward the end of exercise may simply have been the result of the termination of stimulated episodic GH secretion.

We have concluded from these studies that hypohydration ordinarily elicits an exacerbated stress hormone response during exercise in the heat. This observation is consistent with the additional physiological strain imposed by the hypovolemia imposed during hypohydration. Furthermore, heat acclimation attenuates the endocrinological responses to hypohydration and exercise in the heat. Again, this result may be consonant with the hemodilution associated with heat acclimation; presumably, the hypovolemia of hypohydration would be at least partially counter balanced by the acclimation effects. In fact, our data (19) indicated on 11% and 5% reduction in plasma volume at rest when hypohydrated before and after heat acclimation, respectively. Using the magnitude of the PC and GH responses as a metric for assessing relative strain imposed, the hot-wet and hot-dry conditions simulated in the current experiment were approximately of equivalent stress intensity. Supplementary experiments are contemplated using alternative methods to achieve hypohydration, and considering also the effects of other hypohydration levels as well as hyperhydration.

Acknowledgements and Disclaimers

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The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USARMRDC Regulation 70-25 on Use of Volunteers in Research.

Figure Legend

Fig. 1. Effects of hypohydration and acclimation on plasma levels of cortisol during exercise in a thermoneutral environment. Mean values $\pm$ SEM are depicted for n = 16 in all cases except n = 15 pre-acclimation, hypohydration, times 2 and 4. Blood samples were removed after standing for 20 min (time 0), 15-20 min of the first and second exercise bouts (times 1 and 2) and at the end of exercise (time 4). Exercise consisted of walking at 1.34 $m \cdot sec^{-1}$, level treadmill in an environmental temperature of 20°C and rh = 40%.

Fig. 2. Effects of hypohydration and acclimation on circulating levels of cortisol during exercise in a hot-wet environment. All conditions are as noted under Fig. 1 except Ta = 35°C, rh = 79%, and n = 15, pre-acclimation, euhydrated, time 4 and n = 14, post-acclimation, hypohydrated, time 4.

Fig. 3. Effects of hypohydration and acclimation on plasma cortisol levels during exercise in a hot-dry environment. All conditions are as noted under Fig. 1 except Ta = 49°C, rh = 20% and, n = 14, pre-acclimation, hypohydrated, time 4.

Fig. 4. Effects of hypohydration and acclimation on plasma growth hormone levels during exercise in a thermoneutral environment. All conditions are as noted under Fig. 1.

Fig. 5. Effects of hypohydration and acclimation on circulating growth hormone levels during exercise in a hot-wet environment. All conditions are as noted under Figs. 1 and 2.

Fig. 6. Effects of hypohydration and acclimation on plasma levels of growth hormone during exercise in a hot-dry environment. All conditions are as noted under Figs. 1 and 3.

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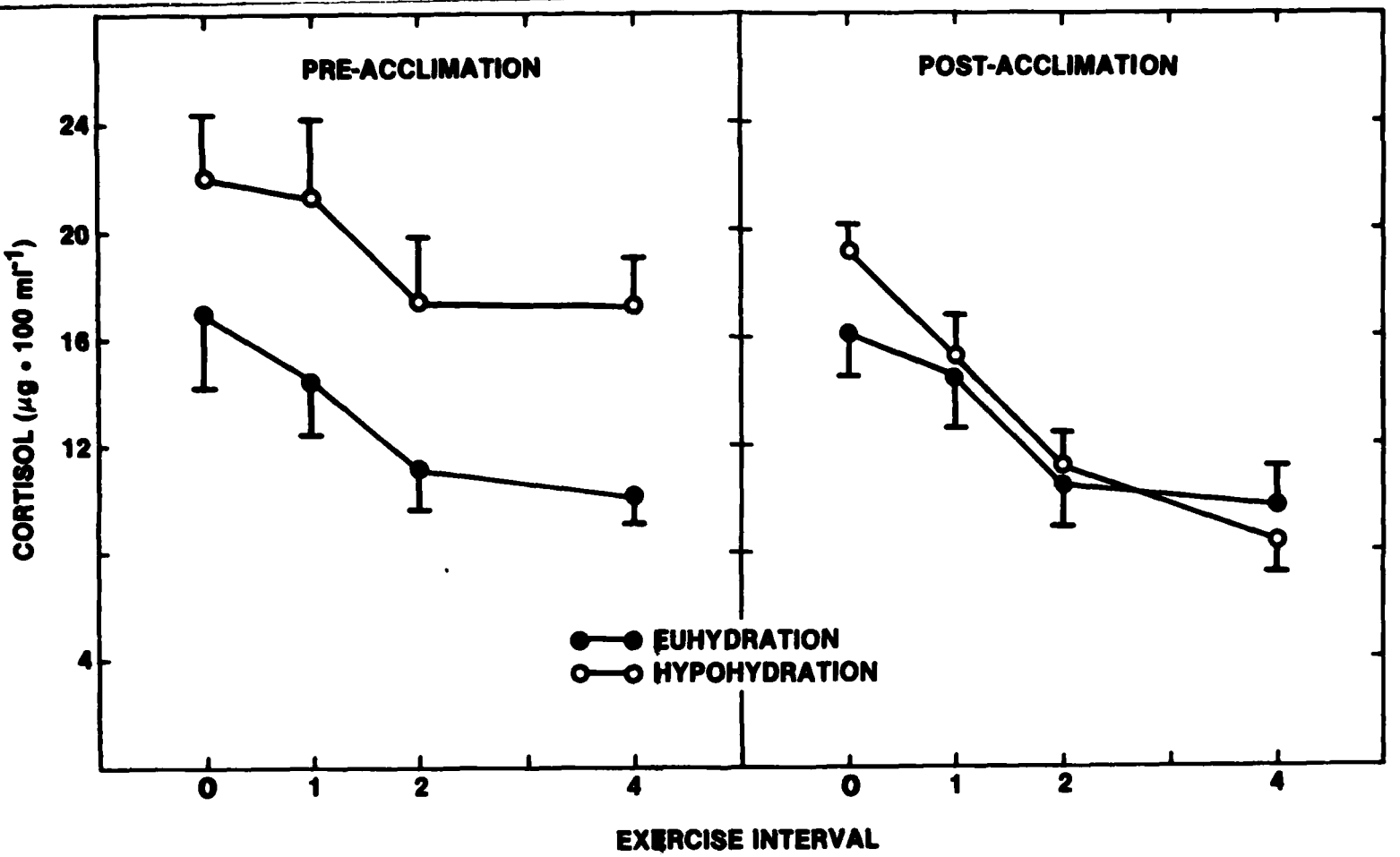
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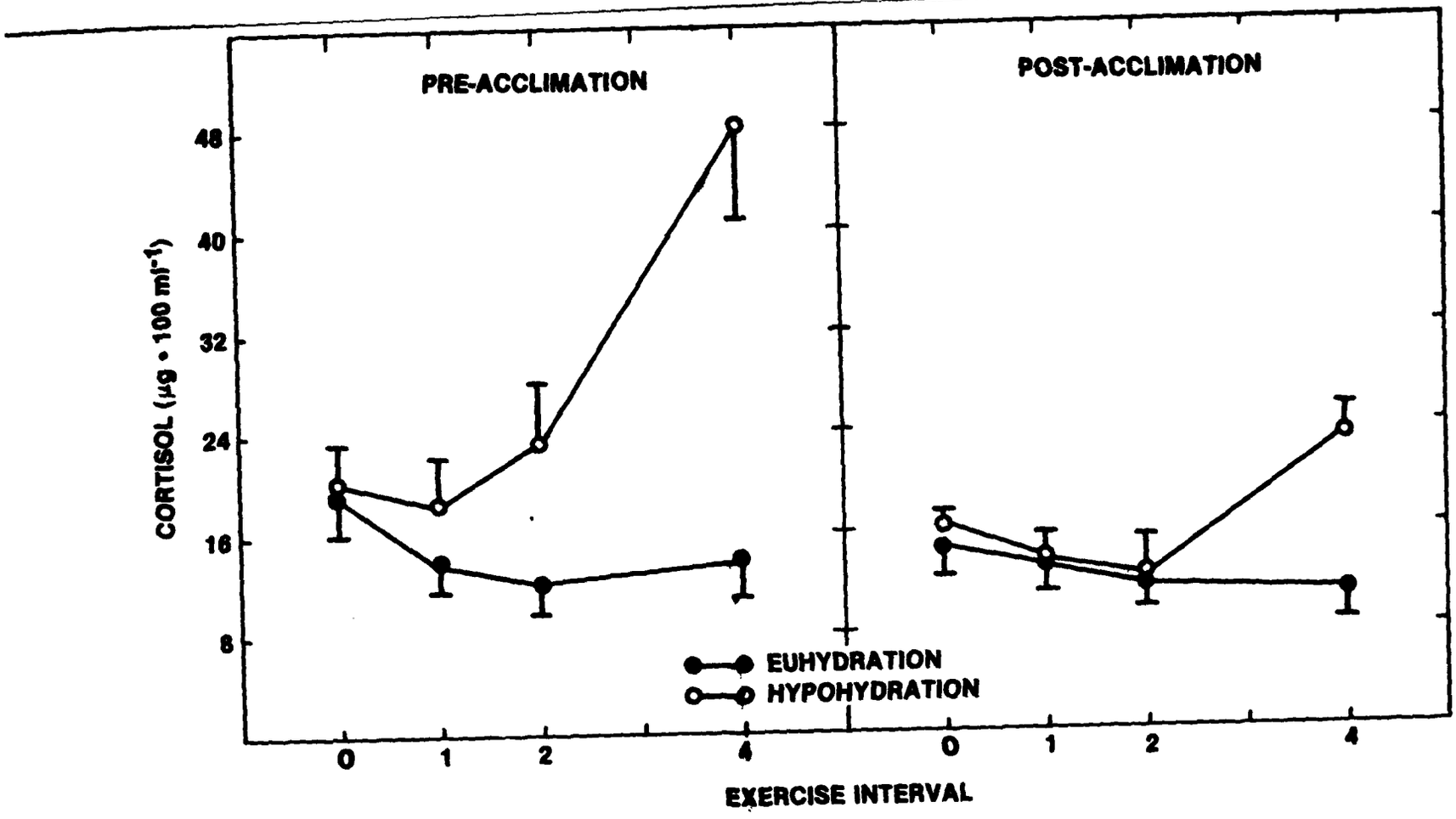
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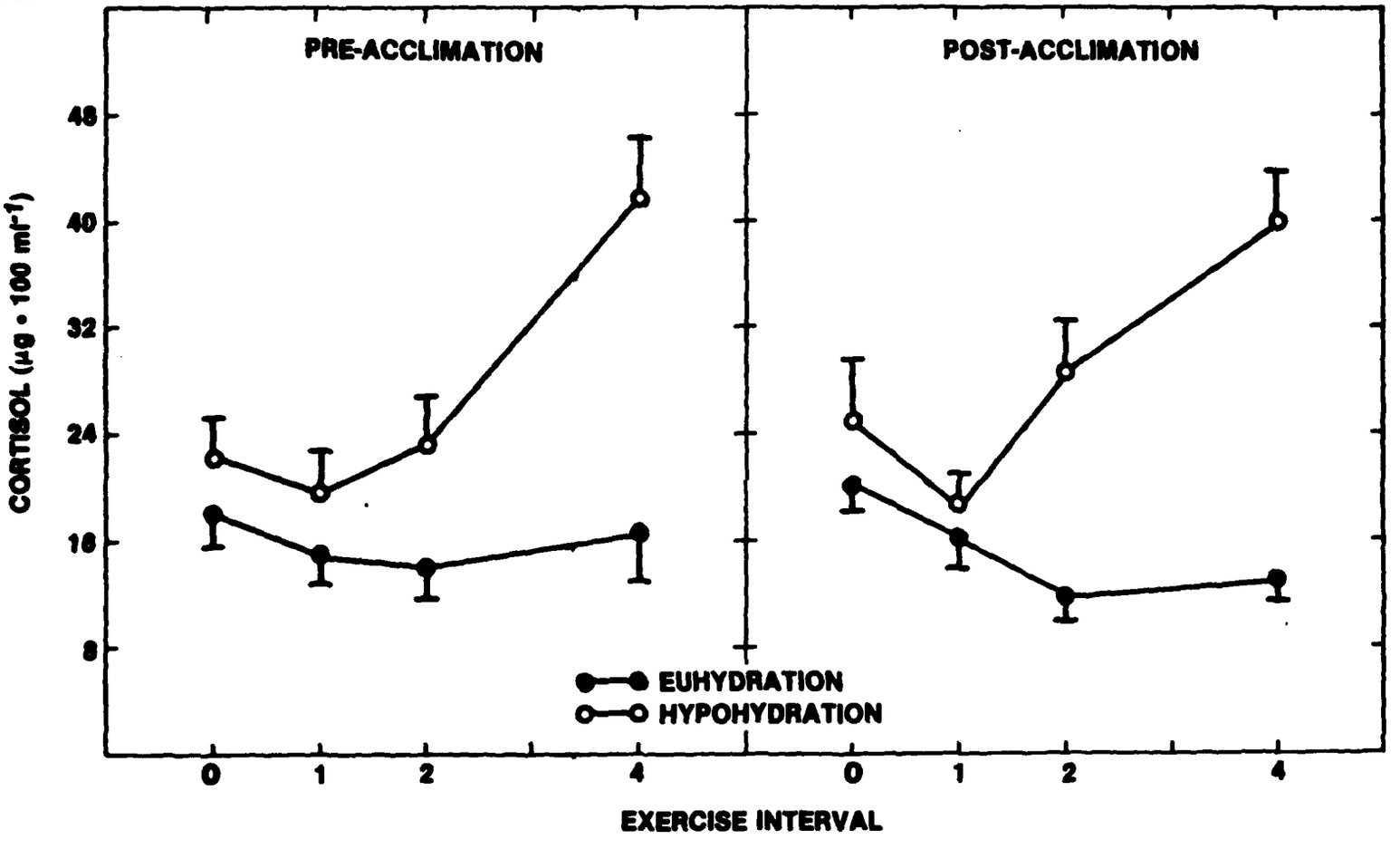
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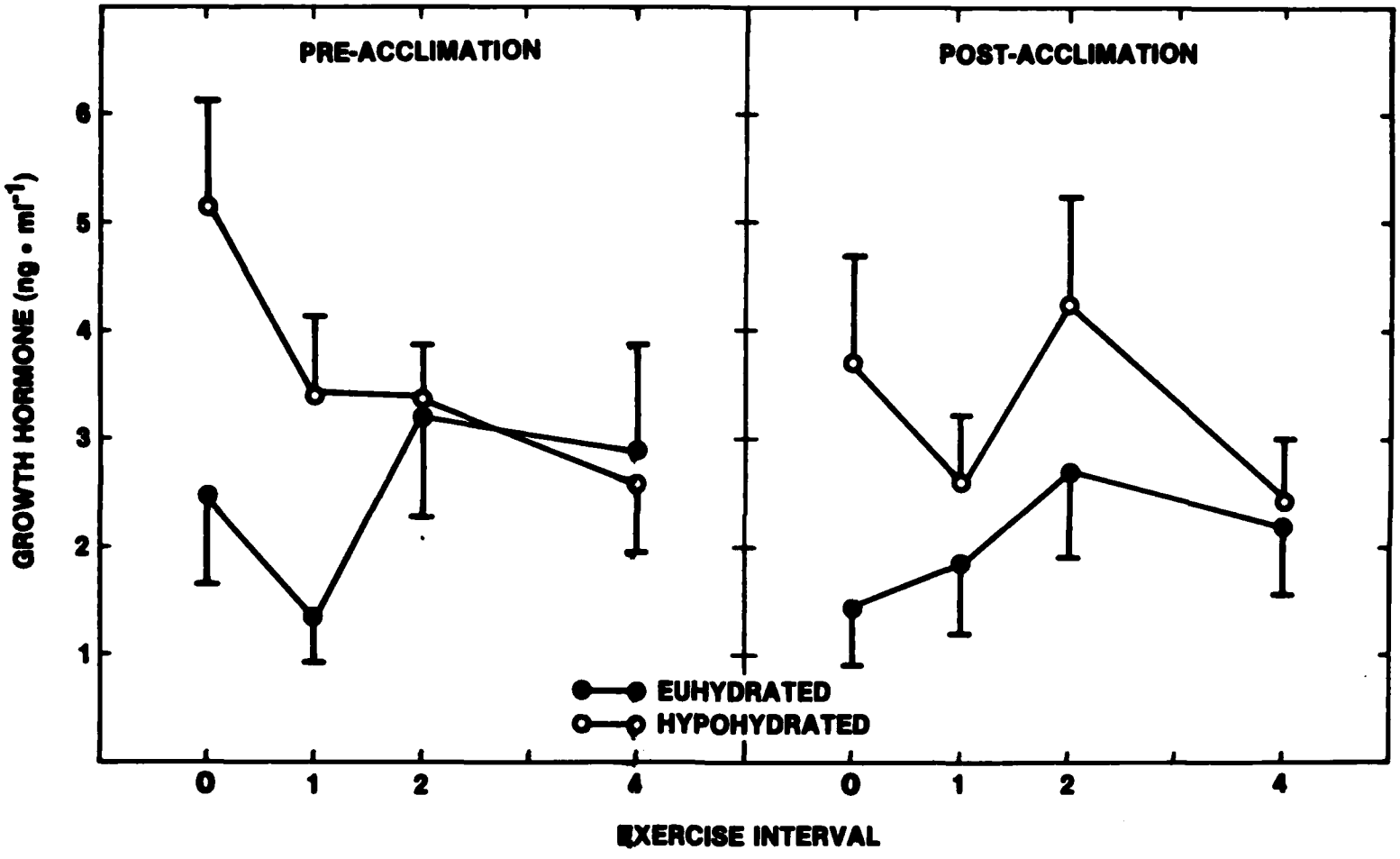
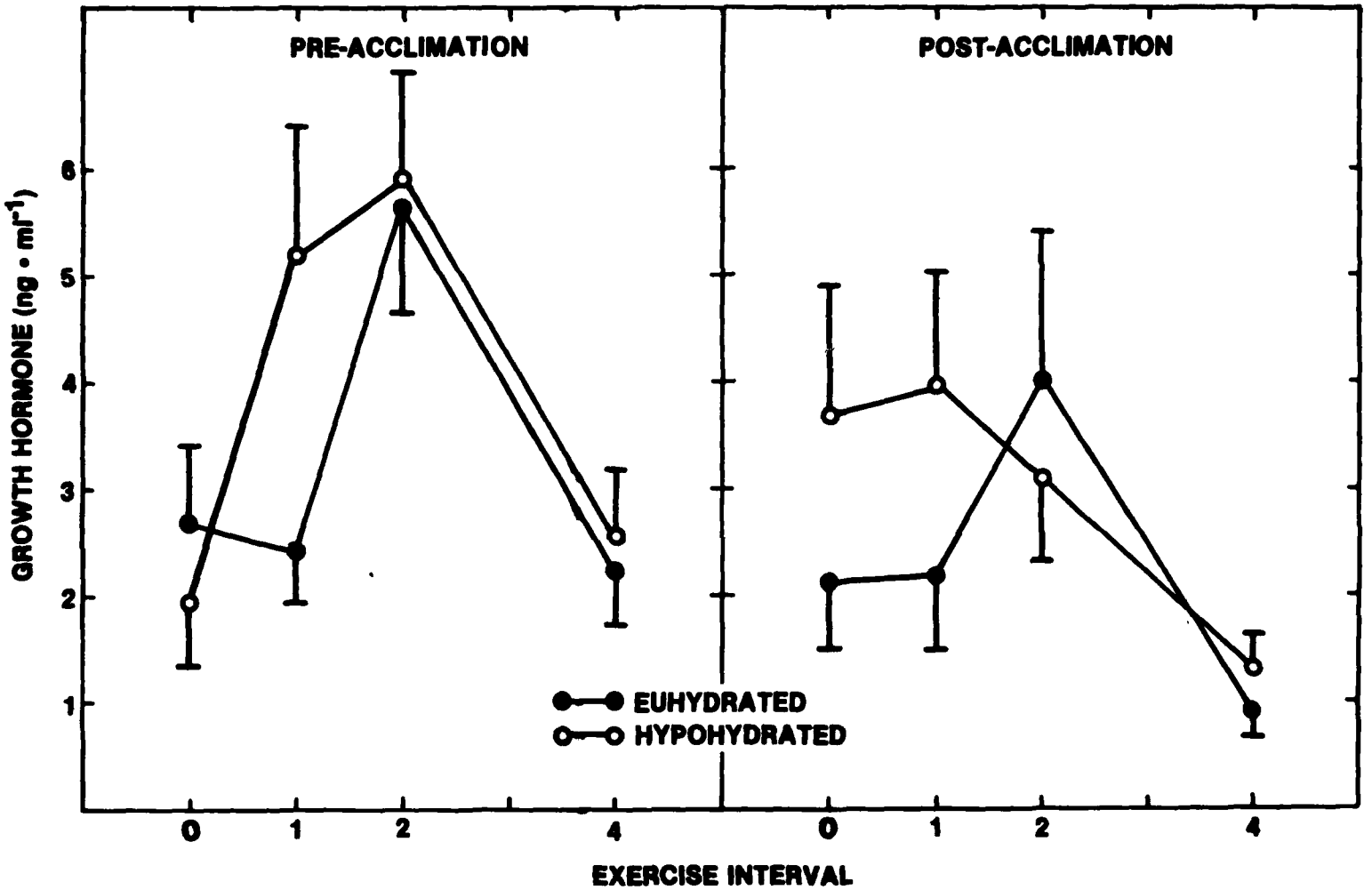
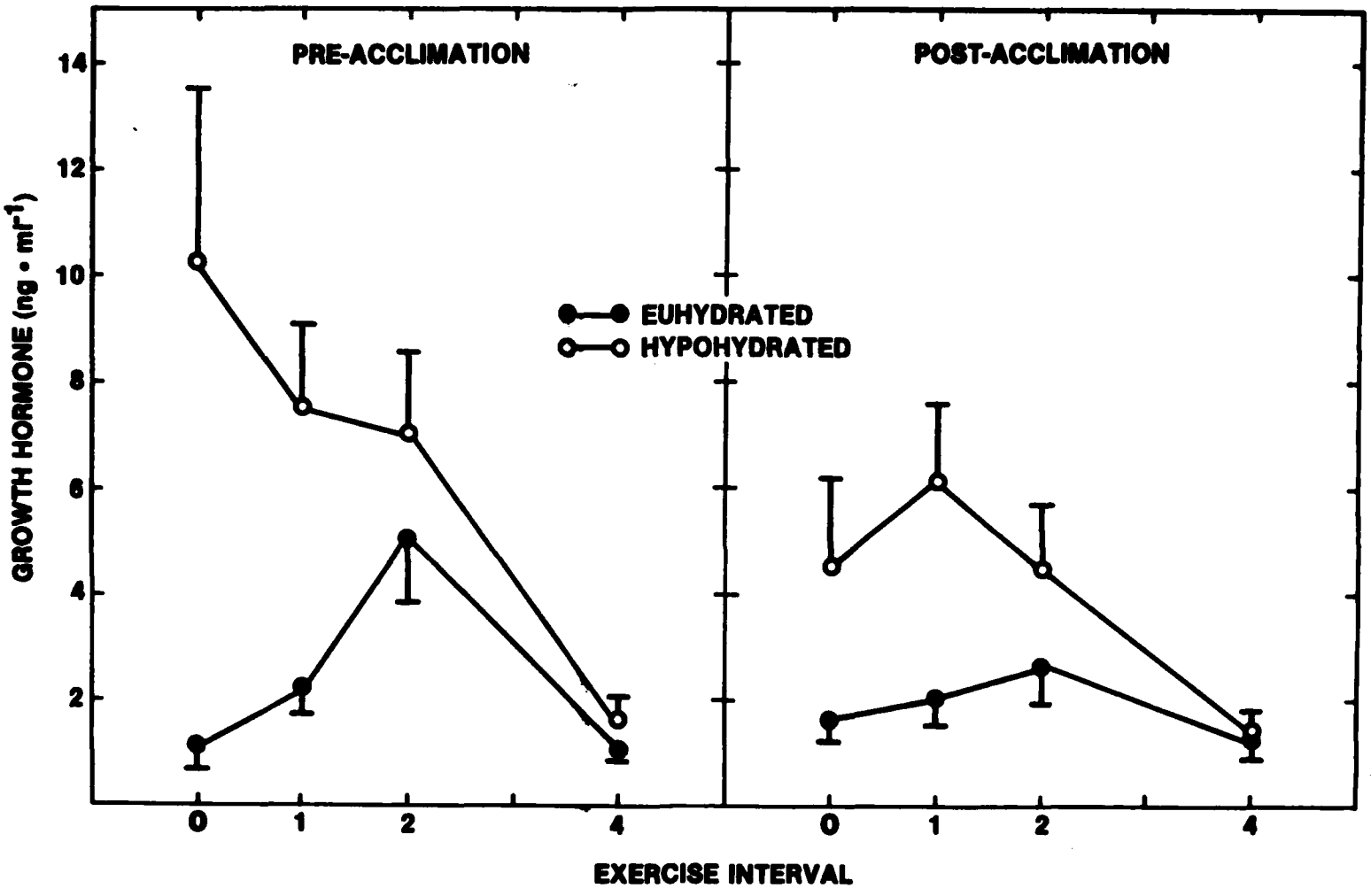
2
4
EXERCISE INTERVAL


PRE-ACCLIMATION
POST-ACCLIMATION
GROWTH HORMONE (ng • ml⁻¹)
6
5
4
3
2
1
EUHYDRATED
HYPOHYDRATED
0
1
2
4
0
1
2
4
EXERCISE INTERVAL


PRE-ACCLIMATION
POST-ACCLIMATION
GROWTH HORMONE (ng • ml⁻¹)
6
5
4
3
2
1
EUHYDRATED
HYPOHYDRATED
0
1
2
4
0
1
2
4
EXERCISE INTERVAL


PRE-ACCLIMATION
POST-ACCLIMATION
EUHYDRATED
HYPOHYDRATED
GROWTH HORMONE (ng • ml⁻¹)
14
12
10
8
6
4
2
0
1
2
4
EXERCISE INTERVAL